

Professional comments—the relevance of measuring salt intake

Motivated patients may not need to measure their intake

Many motivated patients can follow the dietary guideline on salt consistently and get excellent results without needing to measure their salt intake. Measurement is essential for sceptics who are quite convinced their intake is low.

Measurement is also useful for patients with a disappointing clinical response—it identifies the few who need more help and longer follow-up. Note also that health problems due to salt are not necessarily reversible—a few martyrs have self-sustaining hypertension in spite of good urine results (*Salt Matters*, pages 151–152). This can be demonstrated experimentally—most rats with salt-induced hypertension make at least a partial recovery when the cause is removed, but a few do not [1].

Sceptics do need to measure their intake

Many patients genuinely believe their meals are low in salt—a Canberra lawyer “never touched the stuff after hearing it was harmful”. They are unaware that table salt and cooking salt account for only 15% of the average person’s total salt intake. Today only 10% is the natural salt content of the unsalted foods our ancestors ate throughout human evolution, and the remaining 75% is the salt we never see—the unnecessarily heavy load of excess salt hidden from sight in processed foods [2].

All sceptics should be invited to measure their salt intake accurately with a 24-hour urine collection. You can then tell them the intake that usually has a clinical result (for example sodium excretion below 50 mmol/day prevents fluid retention [3]. For preventing the vertigo of Meniere’s disorder this is usually “more effective and less troublesome than diuretics” [4].

Monitor dietary compliance with follow-up 24-hour urine collections. Only 24-hour collections can give results accurate enough for clinical practice, as “spot” urine samples show unacceptable fluctuation due to the circadian rhythm in sodium excretion.

The Heart Foundation’s recommendation to measure salt intake with 24-hour urine collections [5] makes it feasible to manage hypertension at the highly effective DASH-Sodium limit of 65 mmol/day [6, 7].

Even the 24-hour urine collection contains traps that need to be avoided:

- heavy loss of salty sweat can make urinary excretion a much less accurate index of intake;
- however at very low salt intakes sweat is far less salty, with much less salt loss from heavy sweating;
- most people have huge swings in salt *intake* from day to day, reflected in huge swings in sodium *excretion*. This is because a typical diet varies all the time—with a different mix of processed foods—because the sodium content of processed foods ranges from about 5 to about 9000 mg/100g;
- the paradoxical result is that one patient may excrete twice as much sodium as another in a 24-hour urine collection, even if both have the same *habitual* (average) salt intake;
- people who dislike taking a large bottle to work for collecting urine may insist on weekend collections, with possible disadvantages—different meals (missing breakfast) and sometimes drinking beer all day Saturday with very little food and hence misleadingly low sodium excretion;
- women retain sodium during the premenstrual week, especially those with premenstrual syndrome (PMS), and excrete less of their intake than usual. They should preferably start a urine collection a week or 10 days after a period finishes;

- a common mistake is over-collection (fully explained in the patient handout);
- another common mistake is under-collection (again see the patient handout);
- there is no fool-proof test of completeness of collection (see the section below on Interpretation of the report) [8].

“Spot” urines and 8-hour overnight urines are claimed to be accurate enough for comparisons of population averages, but it may be worth repeating that for clinical use the Heart Foundation recommends only 24-hour urine collection [5], as there is no other way to know if the individual is living safely within the 65 mmol/day limit needed to expect the DASH-Sodium results. DASH-Sodium obtained its landmark results with unprecedented accuracy by supplying all the food and verifying the sodium level with 24-hour urine collections [7].

The pathology request form

The lab is preferably asked to report the 24-hour sodium, potassium and creatinine excretion. Creatinine is used as an index of completeness of collection (not for creatinine clearance).

Some urban practices send the patient to the lab to collect the container, but this is always inconvenient, and the lab can keep urban practices supplied with a stock of containers. If they are 2-litre containers it is safer to supply two (the second being returned empty if unused). People who admit to a different meal pattern at the weekend should preferably collect urine during the week.

Interpretation of the report

The report will contain the following information:

Volume. The average volume has been put at about 1.5 litres, but there is wide variation depending on the patient’s sex, body size and drinking habits. A small volume under 700 mL suggests under-collection and a large volume over-collection. Men usually pass a larger volume—especially beer drinkers—but one respectable Hobart woman of average build and undoubted sobriety drank enough water to produce 6 litres in 24 hours.

Creatinine. Although there is no proof of completeness of collection, creatinine provides another clue. Creatinine excretion is related to muscle metabolism, varying with body size and habitual activity, and is above average in manual workers, athletes and heavy meat eaters and below average in vegetarians. With these reservations the sodium/creatinine ratio can allow a rough correction for body size and sex difference [9] when judging sodium excretion rate. Another way of correcting for these differences in sodium excretion is to compare the 24-hour Na/K ratio—it reflects the Na/K ratio of the diet, not the meal size.

Potassium. The 24-hour potassium excretion seldom exceeds 100 mmol on the typical Australian diet, but eating more fruit and vegetables can raise it to 150 mmol, and the more potassium the better (provided renal function is normal), up to 200 mmol. One Slow-K tablet supplies 8 mmol and a single banana can supply over 10 mmol.

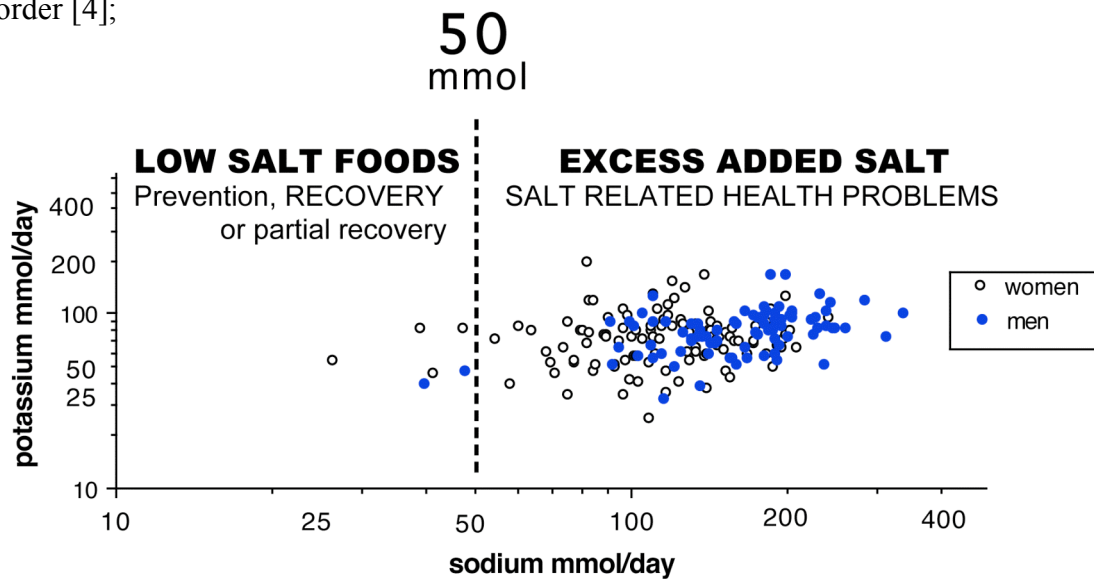
A low potassium excretion rate suggests under-collection or a poor diet, but allowance may be needed for small physique, sedentary habits (small meals) or dieting for weight loss. Polished rice has 70% less potassium than brown rice, giving Chinese food a relative potassium deficiency [10] and white flour, bread and pasta have this problem in our own culture. Faecal potassium loss in chronic diarrhoea and ulcerative colitis reduce urinary potassium excretion relative to intake.

Sodium. Don’t be misled by the typical lab reference range suggesting perhaps that 220 mmol is “normal”. Reference ranges are purely statistical, showing the middle 95% of the distribution in

an industrial society where the diet is artificially loaded with excess salt. The distribution of urinary sodium excretion is abnormal—like that of serum cholesterol [11]—and unrelated to *physiological* normality.

In 1995 a dietary survey recruited 194 people by a systematic selection from the Hobart electoral roll. Before hearing any mention of salt, they made and delivered a 24-hour urine collection, in which the male average sodium excretion was 170 mmol (range 39–337 mmol) and female 118 mmol (range 26–241 mmol). The overall range from 26 to 334 mmol—a more than 12-fold difference—is typical in population surveys, and is the predictably random result of choosing processed foods without reading the label for salt (or sodium) content.

This is a scatter plot of the data showing their sodium and potassium excretion rates in relation to the 50 mmol boundary, above which salt begins to cause salt-related health problems. The first to appear is fluid retention [3], which is a very important trigger for the vertigo of Meniere's disorder [4];



The salt in “normal food”

Patients who feel sure their meals are low in salt will need to be shown this plot and helped to understand it. They may find it hard to believe that all but one of those people assured us they were eating “normal food” (the exception said she was following the Pritikin diet).

The random variation of sodium excretion from under 50 to over 300 mmol/day on a diet of “normal food” is simply what happens when nobody looks at food labels. A survey of Chicago business people found greater random variation between four collections by one individual than the variation *between* individuals, and the authors estimated they would need 14 separate urine collections to find a figure within 10% of one person's *habitual* intake [8].

The salt in selected food

Everybody can create order out of this chaos at will—all they have to do is to select every food in every meal for strict compliance with the dietary guideline to choose foods low in salt. The Menzies Research Institute has made the empirical observation that this simple guideline keeps the 24-hour urinary sodium excretion rate permanently below 50 mmol, usually ranging from about 20–40 mmol/day for men and proportionately less for women [12, 13].

Note that six people excreted less than 50 mmol in the Hobart data by pure accident when they thought they were eating “normal food”.

The powerful therapeutic effect of a daily total sodium intake of 1000 mg (43 mmol) is well known, but it has been a very difficult and demanding diet to follow. Now the salt guideline makes it easy enough to bring it within reach of everyone.

The Heart Foundation Guide to management of hypertension, 2008

The Heart Foundation has adopted the 65 mmol target of the DASH-Sodium study, which is only marginally more strict. This too has been a very difficult and demanding diet by any other approach. Now the salt guideline brings it within reach of everyone and allows a small margin for accidental mistakes.

References

1. Dahl LK. Effects of chronic excess salt feeding. Induction of self-sustained hypertension in rats. *Journal of Experimental Medicine*. 1961;114:231–36.
2. James WPT, Ralph A, Sanchez-Castillo CP. The dominance of salt in manufactured food in the sodium intake of affluent societies. *Lancet*. 1987;1:426–29.
3. Freis ED. Salt, volume and the prevention of hypertension. *Circulation*. 1976;53:589–95.
4. Halmagyi GM, Cremer PD. Assessment of dizziness. *J Neurol Neurosurg Psychiatry*. 2000;68:129–34.
5. National Heart Foundation of Australia. Salt and hypertension (professional paper); May 2007.
6. National Heart Foundation of Australia (National Blood Pressure and Vascular Disease Advisory Committee). Guide to management of hypertension 2008. Quick reference guide for health professionals; 2008.
7. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. *New England Journal of Medicine*. 2001;344:3–10.
8. Liu K, Stamler J. Assessment of sodium intake in epidemiological studies on blood pressure. *Annals of Clinical Research*. 1984;16(Suppl43):49–54.
9. Woodward DR, Beard TC, Ball PJ, Hornsby H, von Witt RJ, Dwyer T. Should the male and female RDI for sodium be the same? [abstract]. 16th Dietitians' Association of Australia National Conference; 1997 14–17 May; Hobart, Tasmania; 1997.
10. Beard TC, Liu S, Wang TY, Wang YG, Dwyer T, Liang LQ. Is it feasible to prevent hypertension and stroke in China? *Australian Journal of Nutrition and Dietetics*. 1993;50:146–51.
11. Rose G. Sick individuals and sick populations. *International Journal of Epidemiology*. 1985;14:32–8.
12. Beard TC. The dietary guideline with great therapeutic potential. *Australian Journal of Primary Health*. In press.
13. Beard TC. *Salt Matters: the killer condiment*. Sydney: Hachette Livre; 2007.